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Emergence of African swine fever in the North Eastern Region of India

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Abstract

African swine fever (ASF) is a highly contagious haemorrhagic viral disease of domestic and wild pigs. It is responsible for serious economic and production losses due to heavy mortality. The continuing spread of ASF from Africa to Europe and recently to China and other Southeast Asian countries threatens global pork production and food security. It is caused by a large DNA virus of the *Asfarviridae* family. The virus also infects ticks of the genus *Ornithodoros*, which may transmit the disease to susceptible pigs. The disease is currently confined to Africa, Europe and Asia. ASF is first recorded in domestic pigs in the State of Assam and Arunachal Pradesh of NE India in May, 2020. Route of transmission may be direct contact with infected domestic or wild pigs; indirect contact and through ingestion of contaminated feed or garbage. The clinical signs of ASF are similar to classical swine fever (CSF) but the ASF virus is unrelated to the CSF virus (an RNA virus). Acute form of ASF is characterized by high fever and haemorrhages in the skin apart from other common signs and mortality may reach upto 100%. Currently, there is no approved vaccine for ASF, although some encouraging progresses have been made in this direction. Besides, proper hygiene and strict biosecurity measures, the disease have been controlled by mass slaughter of affected pigs and proper disposal of carcasses.

Keywords: African swine fever, emergence, epidemiology, prevention and control

Introduction

African swine fever (ASF) is a highly contagious viral disease that affects domestic and wild pigs typically resulting in an acute haemorrhagic fever and heavy mortality. It is an OIE (World Organisation for Animal Health) listed disease. As the name indicates that the disease was originally confined to Africa, it is believed to have entered Europe for the first time in 1957 when it was introduced into Portugal from West Africa^[1]. The disease has spread to Asia including China (home to half of the world's pig population), in 2018 that led to cullings on a massive scale leading to an increase in the price of pork. The disease was first reported in May, 2020 in India from the North eastern states of Arunachal Pradesh and Assam^[2] causing economic losses to the tune of Rs 60 crore in Assam and ruined any prospects of establishing the northeastern states as a hub for the export of pork^[3,4]. The disease is transmitted by both direct and indirect means of transmission including biological vector like soft ticks. ASF causes a spectrum of disease manifestations, from highly lethal to subclinical, depending on host characteristics and pathogenic potential of the virus strain^[5]. The clinicopathological condition of ASF is similar to classical swine fever (CSF), which is caused by a different virus, and they are differentiated by laboratory testing^[2]. Currently, there is no preventive vaccine against ASF. Hence, the disease is controlled by massive culling and hygienic and biosecurity measures.

Etiology

African swine fever virus (ASFV) is a large enveloped double-stranded DNA virus, which is the sole member of the genus *Asfivirus* within the family^[6]. It is the only known DNA arbovirus and is transmitted by soft ticks. The genome varies in length between approximately 170 and 193 kbp, mainly due to loss or gain of multigene family members. ASFV replicates in the cytoplasm of host cell and contain all the essential genes required for the cytoplasmic replication^[7].

Many ASFV genes inhibit host defenses, including type I interferon, the main host antiviral pathway, and programmed cell death, or apoptosis. There is very low mutation rate in ASFV DNA due to accurate proof reading of DNA polymerase and virus-encoded base excision DNA repair system. The lack of related viruses means recombination with other viruses is very unlikely. Thus, the risk of ASFV jumping a species barrier is considered to be negligible.

Five different genotypes of ASFV by restriction endonuclease of viral DNA have been described [8]. All European and south American isolates are within one group and the African shows greater variation. However, 24 genotypes have been described based on partial p72 gene sequence for major capsid protein [9]. There are also variations in virulence, some strains cause severe disease with near 100% mortality, others cause transient disease or inapparent infections.

ASFV are stable in the environment at low temperature and heat inactivated at 56°C for 70 min (60°C for 20 min). It is susceptible to ether and chloroform, inactivated by 8/1000 sodium hydroxide (30 min), hypochlorites-2.3% chlorine (3 min), 3/1000 formalin (30 min), 3% orthophenylphenol (30 min) and iodine compounds [10]. The virus remains viable for long period in blood, faeces, tissues, especially uncooked or undercooked pork products.

Epidemiology

Distribution of the disease

ASF was first described in 1921 in Kenya [11] and since then major outbreaks in Africa are regularly reported. The disease remained restricted to Africa until 1957, when it was reported in Portugal. From there, it spread to Spain and France and they were able to control the disease by the mid-1990s through a slaughter policy [1]. In 1971, an outbreak of the disease occurred in Cuba, resulted in the slaughter of around 5 lakhs pigs to control the disease [12]. The ASFV also crossed the Atlantic Ocean, and outbreaks were reported in some Caribbean islands, including Dominican Republic and Haiti. The genotype II of ASFV was introduced into Georgia in 2007, which spread throughout the Caucasus, into the Islamic Republic of Iran, the Russian Federation and, in July 2012, into Ukraine [13]. Wholesale buyers, particularly the military food supply system, having been implicated multiple times in the illegal distribution of contaminated meat were vectors for the virus's spread according to the FAO report.

The disease was recorded in few other countries like Belarus Lithuania, Poland, Latvia and Estonia during 2013-2015 [14, 15, 16]. Similarly, in Europe, many countries reported the first occurrence of the disease since 2016. Moldova notified the disease as present in September 2016, then Czech Republic in June 2017, followed by Romania in July 2017, Hungary in April 2018, Bulgaria in August 2018, the recurrence of the disease was reported by Belgium in September 2018 (last event occurred in 1985) [2].

The virus has also spread to Asia in 2018, affecting more than 10 percent of the total pig population in several countries, leading to severe economic losses in the pig sector [17] and outbreaks have been reported continuously thereafter. In July 2019, authorities announced the first outbreak of African swine fever in Slovakia [18]. In February 2020, authorities announced the first outbreak of African swine fever in a restricted area of Northern Greece [19].

On May 2020, the first ASF disease outbreak was reported in the state of Assam and Arunachal Pradesh in India [2]. According to the state governments as many as 17,118 pigs in 422 villages of Lakhimpur, Dhemaji, Biswanath, Sivasagar,

Jorhat, Dibrugarh, Majuli, Golaghat, Nagaon, Kamrup (Metro), Morigaon, Karbi Anglong, West Karbi Anglong and Sonitpur districts of Assam and over 4,500 in Arunachal Pradesh so far died due to ASF [3, 4]. It is speculated that the virus has entered Assam through transboundary transmission from China through Tibet. Sale and consumption of pork meat have been banned in the affected districts of Assam.

During 2016 to June, 2020 total outbreaks of ASF 128, 10559 and 21578 were reported from Africa, Asia and Europe, respectively. African countries reported outbreaks only in swine, Asia mainly in swine, while Europe mainly in wildboar [2].

Transmission

The epidemiology of ASF is complex and varies depending on the environment, pig husbandry, the presence/absence of relevant tick vectors, the presence/absence of wild pigs and human behavior. The virus is found in tissues and body fluids of infected pigs. Pigs usually become infected by direct contact with infected pigs or by ingestion of garbage containing unprocessed infected pig meat or pig meat products [20, 21]. Biting flies and ticks, contaminated premises, vehicles, equipment or clothing can also spread the virus to susceptible animals [22]. The warthog (*Phacochoerus africanus*) and giant forest hog (*Hylochoerus meinertzhageni*) can serve as natural reservoir of the virus without any sign of clinical disease [23]. Spread from this reservoir is via the soft tick under the genus *Ornithodoros*. The tick will ingest the virus when taking a blood meal and then pass it on when feeding on susceptible animals. Incidentally, no soft tick has been recorded in the NE region of India. The ASF-infected meat (including frozen or salted products) can transmit the virus over long distances (thousand km) through the market chain. ASF is not a public health threat or a food safety concern. Details on the epidemiology of ASF have recently been reviewed [24].

Pathogenesis

ASF is characterized by severe leukopenia, mostly associated with lymphopenia leading to a general state of immunodeficiency. Initially, the virus enters the pigs following an oral-nasal route or after the bite of an infected soft tick. Initially, the virus replicates in the tonsils or regional lymph nodes, spreading through the lymph and blood to secondary organs of replication (lymph node, bone marrow, spleen, lung liver and kidney) within 2–3 days, and then spreading to the rest of the organs, where virus can replicate in a variety of cells [25].

ASF is characterized by a massive destruction of the lymphoid organs and tissues, including spleen, lymph nodes, thymus, and tonsils [26]. There are a large proportion of B and T lymphocytes and macrophages undergoing cell death in acute ASFV infection. ASFV does appear to replicate in T and lymphocytes and the associated lymphopenia is thought to be due to apoptosis of lymphocytes and necrosis of lymphoid organs [27]. Monocytes and macrophages are the main target for ASFV [28]. The infected monocyte-macrophage appears swollen, with margination of the nuclear chromatin and reveals an intracytoplasmic juxtanuclear inclusion body, The virus replication induce necrosis in the infected cells and virions are released by budding, and can be observed free in the blood, lymph, and the interstitial tissue [25].

Among the typical vascular changes observed in acute ASF, include petechial and ecchymotic hemorrhages in multiple

organs, hemorrhagic, or hyperaemic splenomegaly and pulmonary oedema. In subacute ASF, we can also observe these vascular changes together with a more marked oedema, ascites, and hydropericardium [25]. The most typical lesion in ASF is the hemorrhagic or hyperaemic splenomegaly [29]. Hemorrhages are very common in the late phases of the disease, mostly in organs without a fixed vascular macrophage population, as the renal and gastrohepatic lymph nodes or the kidney [30]. The pathogenesis of haemorrhages appears to be largely related to the development of disseminated intravascular coagulation and destruction of megakaryocytes [31].

Clinical signs

Clinical signs and mortality rates can vary according to the virulence of the virus and the type/species of pig: In an outbreak, all age groups of pigs are affected. The disease may be expressed in peracute, acute, subacute or chronic forms. In peracute cases pigs may die suddenly without any clinical signs. Acute forms of ASF are characterized by high fever, depression, anorexia and loss of appetite, haemorrhages in the skin (visible only on pale skinned pigs) with patches appearing on the tips of ears, tail, feet, chest, or under the belly, laboured breathing, swollen red eyes, eye discharge, vomiting, diarrhea, abortion in pregnant sows and death within 4-15 days and mortality rates may be as high as 100% [32]. Wild boars (*Sus scrofa*) show the same acute signs of disease as domestic pigs. Subacute and chronic forms are caused by moderately or low virulent viruses, which produce less intense clinical signs that can be expressed for much longer periods. Mortality rates are lower, but can still range from 30-70%. Chronic disease symptoms include loss of weight, intermittent fever, respiratory signs, chronic skin ulcers, arthritis and abortion [33]. This form is also caused by moderately or low virulent viruses and develops over 2-15 months with low mortality. Animals that recover from disease may remain infected for several months or throughout life [10, 34].

Diagnosis

Presumptive diagnosis of ASF can be made on the basis of mortality pattern, clinical signs and post-mortem examination in pigs vaccinated against CSF. However, in countries free from ASF, it cannot be differentiated from CSF by either clinical or post-mortem examination. Bacterial septicaemias (eg., erysipelas and salmonellosis) may also be confused with ASF and CSF. Laboratory tests are essential to distinguish between these diseases [35]. The laboratory diagnosis must be directed towards isolation of the virus by the inoculation of pig leukocyte or bone marrow cultures, the detection of genomic DNA by polymerase chain reaction (PCR) or the detection of antigen in smears or cryostat sections of tissues by direct fluorescent antibody test (FAT) and haemadsorption. Suitable samples include, blood, serum, tonsil, spleen, kidney and lymph nodes. Currently, the PCR is the most sensitive technique and can detect ASFV DNA from a very early stage of infection in tissues, EDTA-blood and serum samples. The PCR is particularly useful if samples submitted are unsuitable for virus isolation and antigen detection because they have undergone putrefaction [36]. Pigs that have recovered from acute, subacute or chronic infections usually exhibit a viraemia for several weeks making the PCR test a very useful tool for the detection of ASFV DNA in pigs infected with low or moderately virulent strains. Virus isolation by the inoculation of pig leukocyte or bone marrow cultures and

identification by haemadsorption tests (HAD) are recommended as a confirmatory test when ASF is positive by other methods, particularly in the event of a primary outbreak or a case of ASF [37]. A challenge experiment involving of suspect material into pigs vaccinated against CSF and unvaccinated pigs may also be used for differentiation of CSF and ASF. As no vaccine is available, the presence of ASFV antibodies is indicative of previous infection and, as antibodies are produced from the first week of infection and persist for long periods, they are a good marker for the diagnosis of the disease, particularly in subacute and chronic forms.

Prevention and control

The control of African swine fever (ASF) has been hampered by the unavailability of vaccines. Therefore, prevention in countries free of the disease depends on execution of appropriate import policies and strict biosecurity measures, ensuring that neither infected pork products nor live pigs are introduced there from the ASF affected countries/areas. The preventive measure may include proper disposal of waste food/feed from ships, aircraft or vehicles coming from affected countries and preventing illegal imports of live pigs and pork products from affected countries.

Proper sanitary measures may be employed including early detection and mass culling through humane slaughter of animals (with proper disposal of carcasses and waste); thorough cleansing and disinfection; zoning/compartmentalisation and movement controls; and detailed epidemiological investigation; strict biosecurity measures on farms during an outbreak and in endemic areas. As observed in Europe and in some regions of Asia, the transmission of ASF seems to depend largely on the wild boar population density and their interaction with low-biosecurity pig production systems. The good knowledge and management of the wild boar population and a good coordination among the Veterinary Services, wildlife and forestry authorities are required to successfully prevent and control ASF [2]. Depending on the epidemiological situation, the involvement of the soft tick vector should also be considered in the control programme.

Regarding vaccination, significant progresses have been made by researchers. Several live attenuated ASFV vaccine candidates, produced by passage in cell culture [38] induce good protection but cause unacceptable adverse clinical reactions, including a chronic form of disease in some vaccinated pigs. Increasing knowledge of the functions of ASFV-encoded genes has opened a route for targeted gene deletions to produce rationally attenuated ASFV vaccines. First report on attenuation to a highly virulent ASFV isolate after deletion of members of MGF360 (multigene family 360) and MGF505 came in 2015 [39]. The deletion mutant i.e. ASFV-G- Δ MGF is also able to confer protection against challenge with the highly virulent ASFV Georgia (ASF-G) isolate. The same group also reported development of putative vaccine ASFV-G- Δ 9GL/ Δ UK (a deletion mutant of ASFV) to be the first rationally designed experimental ASFV vaccine that protects against the highly virulent ASFV Georgia 2007 isolate as early as 2 weeks postvaccination [40]. They also reported development of I177L gene deleted mutant of highly virulent ASFV Georgia isolate (ASFV-G) ASFV-G- Δ I177L that produced a strong virus-specific antibody response and, completely protected the animals when challenged with the virulent parental ASFV-G [41]. Similarly, a subunit vaccine containing eight different genes ASFV genes when delivered

to pigs using an adenovirus prime and modified vaccinia Ankara boost protect pigs against a fatal disease caused by a genotype I ASFV strain ^[42].

Conclusion

ASF is a highly contagious disease causing heavy mortality in pigs. It has spread to Europe and Asia including NE region of India from its original place of Africa and become endemic in many of the countries. No preventive vaccine is available against ASF, therefore, mass culling with classic sanitary and biosecurity measures are the only means of prevention and control of the disease. However, significant progresses have been made towards the development of an effective vaccine.

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